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Health and Environmental Effects Profile for Triallate

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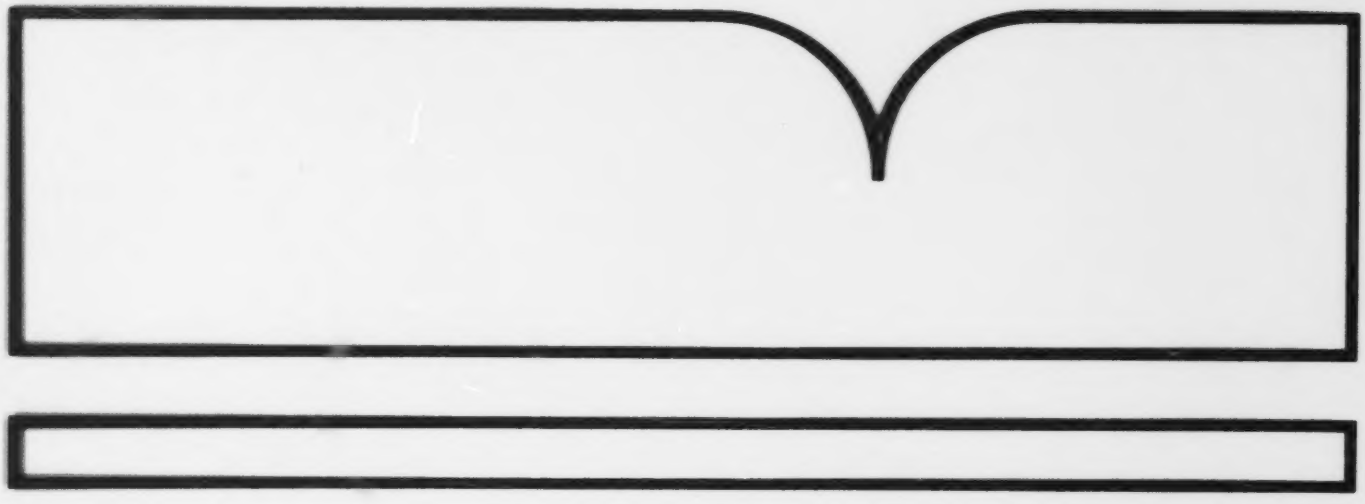

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR TRIALLATE

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16. ABSTRACT

The Health and Environmental Effects Profile for Triallate was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Triallate has been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for Triallate is 1.5×10^{-2} mg/kg bw/day for oral exposure.

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents.The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

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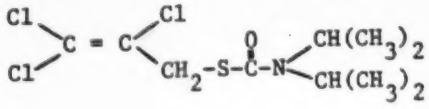
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
CHO	Chinese hamster ovary
CNS	Central nervous system
DNA	Deoxyribonucleic acid
i.p.	Intraperitoneal
LD_{50}	Dose lethal to 50% of recipients
LOEL	Lowest-observed-effect level
MTD	Maximum tolerated dose
NOAEL	No-observed-adverse-effect level
ppm	Parts per million
RNA	Ribonucleic acid
SCE	Sister-chromatid exchange

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Triallate is the common name for S-2,3,3-trichloroallyl diisopropylthiocarbamate. It is also known as bis(1-methyl ethyl)carbamothionic acid, S-(2,3,3-trichloro-2-propenyl)ester; trichloroallyl diisopropyl thiocarbamate; and 2,3,3-trichloro-2-propene-1-thiol-diisopropyl carbamate. This compound was introduced in the market under the code number "CP23426" and has trade names of "Avadex BW" and "Fargo" (BCPC, 1977). The structure of this compound is given below:



Molecular formula: $C_{10}H_{16}Cl_3NOS$

Molecular weight: 304.7

The Chemical Abstract Service (CAS) Registry number for this compound is 2303-17-5.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Triallate is an oily liquid at ambient temperature. Some of the physical properties of triallate are given below (BCPC, 1977; Grover et al., 1978):

Melting Point:	29-30°C
Boiling Point:	148-149°C at 9 mm of Hg
Vapor Density at 25°C:	3.84×10^{-6} g/ℓ
Vapor Pressure at 25°C:	2.07×10^{-4} mm of Hg
Evaporation rate at 25°C:	5.7 µg/cm² hr
Solubility in water at 25°C	4 mg/ℓ

1.3. PRODUCTION DATA

The compound is made by the condensation of diisopropylamine, carbonyl sulfide and 1,1,2,3-tetrachloroprop-1-ene in the presence of a base (BCPC, 1977). Triallate is produced by Monsanto Co. at Lubing, LA (SRI, 1982). Current United States production data are unavailable, but in 1971 the estimated yearly production of this compound was ~1 million pounds (Lawless et al., 1972). From usage data in California and Minnesota, Von Rumker et al. (1974) estimated that $\geq$72,000 pounds of this compound were used in the United States in 1972.

1.4. USE DATA

Triallate is used for the control of wild oats and other weeds in a variety of cereal and other crops (i.e., wheat, barley, oil seed crops, flax and peas) (Cullimore and Smith, 1972; Smith, 1969; Smith and Hayden, 1982a).

2. ENVIRONMENTAL FATE AND TRANSPORT

The two important routes of entry of this herbicide into the atmosphere are spray drift during application to soil and evaporation from soil after application. Information pertaining to the fate and transport of this chemical in the atmosphere is not available. The fate of triallate in the air is likely to be determined by its ability to undergo reactions with O_3, NO_x and free radicals (e.g., RO•, RO_2• and O•) and by its ability to undergo photochemical reactions. In the absence of any qualitative or quantitative data pertaining to such reactions, it is difficult to predict the atmospheric fate of this compound.

Although no comprehensive study relating to the transport of triallate in the atmosphere is available, some theoretical predictions can be made. Because triallate has a moderately high soil adsorption coefficient (Kenaga, 1980; Grover et al., 1979), and because at least some of it is strongly bound to soil (Anderson and Domsch, 1980), it probably will exist in the atmosphere in the particulate-sorbed phase. Both wet and dry deposition are likely to remove particulate-sorbed triallate from the atmosphere.

Little information pertaining to the fate and transport of triallate in aquatic media is available. Chemically, triallate could react with the available free radicals in the aquatic media and/or undergo photochemical reactions. It could also undergo hydrolytic reactions. No information is available regarding the reactivity of triallate with free radicals and light of wavelength >290 nm. The hydrolysis of triallate was studied by Smith (1969). In aqueous solution at pH 4-9 and at a temperature of 25°C, only 10-15% of triallate underwent hydrolysis in 24 weeks. Therefore, hydrolysis is not expected to be a significant fate determining process for this chemical in aquatic media.

Biodegradation is likely to be important in determining the fate of triallate in aquatic media. This speculation may be justified by the well-established biodegradation potential of this compound in soils (Smith and Fitzpatrick, 1970; Anderson and Domsch, 1980), although the biodegradation in ambient aquatic media has not been studied.

The volatility data for triallate from aquatic media are not available. However, based on the vapor pressure and water solubility data for this compound and a comparison of the water evaporation data of diallate (Nassar and Ebing, 1979), the predicted evaporative half-life of triallate is several days. The adsorption of triallate from aqueous solution by two different soils was determined to be >95% (Smith and Fitzpatrick, 1970). This result and the investigations of other researchers (Kenaga, 1980; Grover et al., 1979; Cullimore and Smith, 1972) indicate that sorption on suspended particulate matter with subsequent partial precipitation to the sediment may play a significant role in determining the transport of triallate in aquatic media.

No experimental data are available regarding the bioconcentration of this chemical by aquatic organisms. From binary regression equations, Kenaga (1980) predicted a BCF of 150-280 for triallate. Therefore, it is not expected to be significantly bioaccumulated in aquatic organisms.

The fate and transport of triallate in soils has been studied extensively. Although triallate may hydrolyze faster than diallate in NaOH solution (Smith and Fitzpatrick, 1970), the very slow hydrolysis of triallate in aqueous solution between pH 4 and 8 indicates that triallate is not likely to undergo significant hydrolysis in moist soils. The significance of free radical reactions of triallate in soils has not been investigated. Although the rate of photolysis of triallate in soil has not been studied, based on

the photolysis experiments of Nassar and Ebing (1979) with diallate, some photolysis of triallate present on the surface layer of soil seems likely. The rate of photodecomposition, however, is expected to be slow (Beestman and Deming, 1976). The experimental results of Smith and Fitzpatrick (1970) with two different types of soil also suggest that chemical reactions are insignificant compared to biological degradation of triallate in soil.

The biodegradability of triallate in soils has been studied by a number of investigators (Cullimore and Smith, 1972; Smith, 1969; Anderson and Domsch, 1980). The rate of biodegradation appears to depend on both soil type and moisture content of the soil (Smith, 1970). Thus, soils with higher moisture contents tend to biodegrade triallate faster than soils with lower moisture contents. Similarly, the biodegradation rate is dependent on adsorptive capacity and the microbial population (those responsible for degradation) of the soils. Soils with stronger absorptive capacity are likely to be relatively slow in the biodegradation of triallate (Cullimore and Smith, 1972; Anderson and Domsch, 1980). Depending on the type of soil, the half-life of triallate may vary from 5-12 weeks (Smith, 1969; Smith and Fitzpatrick, 1970; Anderson and Domsch, 1980).

Generally, it is assumed that the persistence of one herbicide in soil may be changed in the presence of other pesticides. This is due to the inhibition of the herbicide-degrading organisms in the soil by the added herbicide (Smith and Hayden, 1982a). In the case of triallate, however, other pesticides such as chloramben, triflurabin, dinosel acetate, 2,4-D, thiophanate and tridemorph did not affect its biodegradability in soils (Anderson and Domsch, 1980; Smith, 1979; Smith and Hayden, 1982a). Chlorpyrifos was found to partially inhibit degradability of triallate in soils (Anderson and Domsch, 1980).

The two important processes that may control the transport of triallate in soils are volatilization and leaching. Both these processes are controlled by the adsorption capability of triallate by the soil. Since triallate has a tendency to adsorb strongly to soils (Kenaga, 1980; Grover et al., 1979; Smith, 1970; Smith and Fitzpatrick, 1970), neither evaporation from soils nor leaching from soils is expected to be significant compared to losses of this chemical by biodegradation. For example, the volatility of triallate from soils was found to be negligible by many investigators (Khan, 1973; Smith, 1970). Jury et al. (1980) found only ~4% loss of triallate from soil after 30 days under 50-100% relative humidity conditions, while Beestman and Deming (1976) reported the half-life for evaporation under constant air circulation to be 11-90 days depending upon the soil characteristics.

At the normal field application rate (1.25 lb/acre), trillate did not readily leach from soil. Depending on soil characteristics, only 5-13% of the amount applied could be eluted by 9 inches of water (Smith, 1969). One year after application of triallate to soil, negligible amounts (~2%) of triallate were recovered from 5-10 cm soil depths, indicating minimal leaching (Smith and Hayden, 1976; Smith and Hayden, 1982b; Smith, 1971).

In conclusion, the major routes of loss of triallate from soils in descending order of importance are biodegradation and volatilization (Anderson and Domsch, 1980; Smith and Hayden, 1982a). Both processes are favored by moist soil conditions and are inhibited by dry soil conditions.

3. EXPOSURE

No monitoring data for triallate are available in STORET (see Appendix). The levels of this compound in water and food are not available from published literature. Long-term field studies, in which triallate was applied annually as a pre-emergence herbicide on plots used to grow barley, have not resulted in detectable triallate residues in barley grain (or straw) (Fryer and Kirkland, 1970). Detection limits were 0.025 ppm for barley grain and 0.05 ppm for the straw. These results indicate that triallate residues may not be found in foods.

Grover et al. (1981) determined the levels of triallate in Canadian air. The maximum concentration of triallate was found to be 200 ng/m^3 during the peak spraying season, gradually decreasing to $\leq$20 ng/m^3 by midsummer and falling below the detection limit in early November.

4. COMPOUND DISPOSITION AND PHARMACOKINETICS

4.1. ABSORPTION

Rapid absorption of triallate following oral administration to rats or rabbits was reported in abstracts of two Russian studies. Khokhol'kova and Pestova (1969) administered a single oral dose of triallate of either 1000 mg/kg [maximum tolerated dose (MTD)] or 1471 mg/kg (LD_{50}) to 105 rats (strain and sex not reported), and observed the treated animals for 96 hours post-dosing. Results of blood level monitoring indicated that triallate appeared in the blood within 15 minutes after dosing, and reached maximum levels in 30 minutes. Rapid absorption of triallate from the gastrointestinal tract was reported in rabbits (sex and number not reported) given a single oral dose of 500 mg/kg (Zhavoronkov et al., 1972). Further details regarding the actual levels of triallate observed in blood plasma or the kinetics of absorption were not reported in the available abstracts.

4.2. DISTRIBUTION

Only qualitative descriptions of triallate distribution in rats, rabbits, hens and sheep administered triallate orally were reported in the abstracts of the studies by Khokhol'kova and Pestova (1969) and Zhavoronkov et al. (1972). Further detail regarding quantitative distribution levels and ratios was not provided in these abstracts.

Maximum levels of triallate in the bodies of rats intubated with 1000 or 1471 mg/kg were observed 6-12 hours post-dosing in the lungs, liver, brain and kidneys (Khokhol'kova and Pestova, 1969). Zhavoronkov et al. (1972) administered triallate in single oral doses of 500 mg/kg to rabbits or 300 mg/kg to sheep, and repeated doses of 1/240 of the LD_{50} (actual dose not reported) to hens for 100 days. In treated rabbits, the largest amount of triallate accumulated in the liver, lungs, kidneys and spleen, while lesser

amounts also accumulated in five other unspecified internal organs. By 7 days post-treatment, levels had decreased below detection. In treated sheep slaughtered 6 days after treatment, traces of triallate were found in the meat that had been in cold storage for 78 days after slaughter. No traces of triallate were found in the organs, meat or eggs of treated hens.

4.3. METABOLISM

Marsden and Casida (1982) administered a triallate-corn oil solution at a dose of 100 μmol/kg to four male albino rats via stomach tube, and collected their urine for 24 hours post-treatment. The urine was then analyzed in duplicate for acid recovery of the haloacrylic acids, 2-chloroacrylic acid (0.016 and 0.020%) and trichloroacrylic acid (0.065 and 0.072%); the low percent recovery was expected, due to the highly reactive nature of the haloacrylic acid metabolites. Marsden and Casida (1982) postulated a mechanism for the formation of the haloacrylic acids, whereby S-methylene hydroxylation facilitated the conversion of triallate to haloacrylic acids, presumably via unstable intermediates, trichloroacroleins.

Marsden and Casida (1982) substantiated their *in vivo* findings with an *in vitro* metabolism assay in a rabbit liver microsomal oxidase system. Rabbit liver microsomes were incubated in ~4 mℓ of reaction medium with 10 μg triallate and either 0 or 3.6 μmol NADPH. In the absence of NADPH, <0.002% of the triallate was metabolically converted to haloacrylic acids. In the presence of NADPH, metabolic conversion of triallate to 2-chloroacrylic acid was 0.26 and 0.10%, and to trichloroacrylic acid was 0.46 and 0.28% in assays of two different incubation mixtures. Therefore, the metabolic conversion of triallate to haloacrylic acids *in vitro* is a NADPH-dependent reaction, indicative of microsomal oxidation. From this *in vitro*

study, Marsden and Casida (1982) concluded that the haloacrylic acids are formed metabolically from triallate by the microsomal oxidases, presumably via trichloroacroleins, which are highly reactive. 2,3,3-Trichloroacrolein is a potent mutagen (Rosen et al., 1980). Although 2-chloroacrolein is thought to be an intermediate in the metabolism of the related herbicide diallate, it is not considered to be an intermediate in the metabolism of triallate (Marsden and Casida, 1982; Schuphan and Casida, 1979; Schuphan et al., 1979).

4.4. EXCRETION

Zhavoronkov et al. (1972) reported complete elimination of triallate 7 days after rabbits were administered a single oral dose of 500 mg/kg. Triallate administered as a single oral dose of 1000 or 1471 mg/kg to rats disappeared from the blood within 12-48 hours after administration, and complete elimination from the body was accomplished 1-3 days post-treatment (Khokhol'kova and Pestova, 1969). Further details regarding the excretion of triallate by treated animals were not reported in the available abstracts of these two Russian studies.

5. EFFECTS

5.1. CARCINOGENICITY

In a 2-year carcinogenicity study conducted at Monsanto Laboratories, Johannsen et al. (1977) administered triallate to groups of 50 male and 50 female rats at dietary concentrations of 0, 50, 100 or 200 ppm. Depressed weight gain was observed in females at the 200 ppm level; food consumption data were not reported. For all dose levels tested, there were no gross pathological changes, and no difference in survival, number of animals with tumors, number of tumors or number of animals with malignant neoplasms when compared with corresponding control values. Further details of this study were not reported in the proceedings abstract, and no published paper on this study was found.

5.2. MUTAGENICITY

Many mutagenicity bioassays of triallate have been conducted using a number of test systems. These studies are summarized in Table 5-1. Carere and Morpurgo (1981) have compiled an excellent review of some of these short-term mutagenicity assays.

The assay used most frequently to test the mutagenicity of triallate was the Salmonella typhimurium reverse mutation assay, both with and without a mammalian metabolic activation system (rat liver S-9). Triallate was shown to be mutagenic in tester strains TA100 and TA1535 both with and without metabolic activation (Carere et al., 1978a,b; Sikka and Florcyzk, 1978; DeLorenzo et al., 1976, 1978; Douglas et al., 1981a,b; Sandhu et al., 1981; Kasica et al., 1981; Wildeman and Nazar, 1982; Shiau et al., 1981). Triallate also induced both base-substitution and frameshift mutations, since dose-related increases in revertants were noted for strains TA1535, TA100 and TA98. In the other strains tested, TA1537, TA1538 and TA1536, triallate

TABLE 5-1

Mutagenicity Testing of Triallate

Assay	Indicator Organism	Application	Concentration or Dose	Activation System	Response[a]	Comment	Reference
Reverse mutation	Salmonella typhimurium	spot test	50-300 µg/plate			NC	Shiau et al., 1981
	TA1535			±S9	+		
	TA1536			±S9	-		
	TA1537			±S9	-		
	TA1538			±S9	-		
	TA98			±S9	-		
	TA100			±S9	+		
Reverse mutation	S. typhimurium TA 1535	plate incorporation	10-1000 µg/plate	±S9	±	dose-related at ≥10 µg/plate	Sikka and Florczyk, 1978
	TA1538			±S9	-		
	TA98			±S9	-		
	TA100			±S9	±	dose-related at ≥10 µg/plate	
Reverse mutation	S. typhimurium TA1535	plate incorporation	50-250 µg/plate	±S9	±	dose-related at ≥50 µg/plate	DeLorenzo et al., 1978
	TA1537			±S9	-		
	TA1538			±S9	-		
	TA98			±S9	-		
	TA100			±S9	±	dose-related at ≥50 µg/plate	
Reverse mutation	S. typhimurium TA1535	NR	NR	±S9	±	NC	DeLorenzo et al., 1976
	TA100			±S9	±		
Reverse mutation	S. typhimurium TA1535	spot test	4800 µg/plate	±S9	+	NC	Carere et al., 1978b
	TA1536			±S9	-		
	TA1537			±S9	-		
	TA1538			±S9	-		

TABLE 5-1 (cont.)

Assay	Indicator Organism	Application	Concentration or Dose	Activation System	Response[a]	Comment	Reference
Reverse mutation	S. typhimurium TA1535	plate incorporation	0.005-5.0 mg/plate	±S9	±	dose-related at ≥0.005 mg/plate	Douglas et al., 1981a,b
	TA1537			±S9	=		
	TA1538			±S9	=		
	TA98			±S9	+		
	TA100			±S9	±	dose-related at ≥0.005 mg/plate	
Reverse mutation	S. typhimurium TA98	NR	NR	±S9	±	NC	Sandhu et al., 1981
	TA100			±S9	±		
	TA1535			±S9	±		Kasica et al., 1981
Reverse mutation	S. typhimurium TA98	plate incorporation	100 µg/plate	±S9	±	NC	Wildeman and Nazar, 1982
	TA100			±S9	±		
Reverse mutation	S. typhimurium TA98	plate incorporation	100 µg/plate	±S9,±S14	±	Triallate was sensitive to plant metabolism in vitro. Addition of S14 (plant homogenate) enhanced the toxicity of triallate to both tester strains.	Wildeman and Nazar, 1982
	TA100			±S9,±S14	±		
Reverse mutation	S. typhimurium TA1535	spot test	4800 µg/plate	None	+	NC	Carere et al., 1978a
	TA1536				-		
	TA1537				-		
	TA1538				-		
Reverse mutation	S. typhimurium 8 strains, NR	spot test	~1-5 µl/plate	None	-	NC	Andersen et al., 1972
Reverse mutation	Escherichia coli WP2	NR	NR	None	-	NC	Sandhu et al., 1981 Kasica et al., 1981

TABLE 5-1 (cont.)

Assay	Indicator Organism	Application	Concentration or Dose	Activation System	Response[a]	Comment	Reference
Reverse mutation	bacteriophage T_4	plaque formation *E. coli*	1 µl	None	-	NC	Andersen et al., 1972
Reverse mutation	rII mutant of bacteriophage T_4	plaque formation, *E. coli*	0.04 µl	None	-	NC	Andersen et al., 1972
Reverse mutation	*Saccharomyces cerevisiae* - D7	NR	NR	±S9	=	NC	Sandhu et al., 1981 Kasica et al., 1981
Forward mutation	*Streptomyces coelicolor*	spot test	4800 µg/plate	±S9	+	NC	Carere et al., 1978b
Forward mutation	*S. coelicolor*	spot test	4800 µg/plate	None	+	NC	Carere et al., 1978a
Forward mutation	*Aspergillus nidulans*	NR	NR	NR	+	NC	Morpurgo et al., 1977
Sex-linked recessive lethal	*Drosophila*	NR	NR	None	-	NC	Sandhu et al., 1981 Kasica et al., 1981
Reverse mutation	*Bacillus subtilis* TKJ6321	spot test	50-300 µg/plate	±S9	+	dose-related at ≥50 µg/plate	Shiau et al., 1981
	TKJ5211			±S9	=		
Chromosome aberrations	Chinese hamster ovary (CHO) cells	cell culture	1.0-2.66 x 10^{-4} M	+S9	+	dose-related increase	Douglas et al., 1981a,b
Sister-chromatid exchange (SCE)	CHO cells	cell culture	1.0-2.66 x 10^{-4} M	+S9	+	dose-related increase	Douglas et al., 1981a,b
Alkaline-sensitive single strand DNA breaks	CHO cells	cell culture	1.0-2.66 x 10^{-4} M	+S9	-	determined by sedimentation in alkaline sucrose gradients	Douglas et al., 1981a,b
L5178Y mouse lymphom athymidine kinase	mouse lymphoma cells	cell culture	NR	NR	+	NC	Sandhu et al., 1981 Kasica et al., 1981

TABLE 5-1 (cont.)

Assay	Indicator Organism	Application	Concentration or Dose	Activation System	Response[a]	Comment	Reference
Mitotic recombination	S. cerevisiae D3	NR	NR	±S9	+	NC	Sandhu et al., 1981 Kasica et al., 1981
Mitotic crossing-over	S. cerevisiae D7	NR	NR	±S9	=	NC	Sandhu et al., 1981 Kasica et al., 1981
Mitotic gene conversion	S. cerevisiae - D7	NR	NR	±S9	=	NC	Sandhu et al., 1981 Kasica et al., 1981
Cell survival	S. cerevisiae - D7	NR	NR	±S9	=	NC	Sandhu et al., 1981 Kasica et al., 1981
Unscheduled DNA synthesis	EUE cells[b]	cell culture	NR	None	+	NC	Benigni and Dogliotti, 1980a,b
Increased unscheduled DNA synthesis	EUE cells[b]	cell culture	3.2×10^{-6} to 1.6×10^{-3} M	None	+	dose-related at $\geq 3.2 \times 10^{-6}$ M	Benigni et al., 1982
Inhibition of DNA synthesis	rat thymocytes	cell culture	10-1000 µg/mL	None	+	at 100 µg/mL, 57% inhibition of DNA synthesis	Rocchi et al., 1980
Inhibition of DNA synthesis	human lymphocytes	cell culture	100 µg/mL	None	+	52% inhibition of DNA synthesis	Rocchi et al., 1980
Increased unscheduled DNA synthesis	human lymphocytes	cell culture	100 µg/mL	None	+	5% inhibition of unscheduled DNA synthesis	Rocchi et al., 1980
Dominant lethal	male mice	i.p. injection	200 or 400 mg/kg	None	-	NC	Johannsen et al., 1977

[a]Responses: +, positive; -, negative; =, negative with and without S9 activation system; ±, positive with and negative without S9 activation system

[b]EUE cells are epithelial-like cells derived from skin and muscle explants of human embryo.

NR = Not reported; NC = no comment

did not elicit a positive mutagenic response. These results indicate that triallate induces revertants in Salmonella typhimurium through base pair substitution. It is also mutagenic in the absence of S-9 activation system, indicating that it is a direct-acting mutagen.

Triallate gave negative mutagenic responses in other reverse mutation assays using Escherichia coli WP2 (Sandhu et al., 1981; Kasica et al., 1981), bacteriophage T_4 and an rII mutant of bacteriophage T_4 (Andersen et al., 1972), or Saccharomyces cerevisiae D7 (Sandhu et al., 1981; Kasica et al., 1981) as indicator organisms. Triallate induced forward mutations in assays of Streptomyces coelicolor (Carere et al., 1978a,b) and Aspergillus nidulans (Morpurgo et al., 1977).

When tested in Drosophila, triallate did not induce a significant increase in the mutation frequency of sex-linked recessive lethals (Sandhu et al., 1981; Kasica et al., 1981). These two reports are in the form of meeting abstracts; no data were provided.

Shiau et al. (1981) tested triallate in a growth inhibition assay using Bacillus subtilis strains TKJ6321 and TKJ5211 as the indicator organisms. Triallate was positive in strain TKJ6321 both in the presence and absence of rat liver S-9, while negative results were observed in strain TKJ5211, regardless of the presence or absence of a metabolic activating system.

Douglas et al. (1981a,b) examined the effect of triallate on chromosome aberrations, SCE, DNA damage and cytotoxicity in CHO cells. Dose-related increases were observed in the frequency of chromosomal aberrations, SCEs and cytotoxicity, indicating that triallate is clastogenic in nature.

A positive mutagenic response was obtained for triallate in the L5178Y mouse lymphoma thymidine kinase assay (Sandhu et al., 1981; Kasica et al., 1981). These investigators also reported a significant increase in mitotic recombinations in S. cerevisiae D3, both in the presence and absence of rat liver S-9; however, negative results were observed in several assays using S. cerevisiae D7, regardless of metabolic activation.

Triallate induced dose-related increases of unscheduled DNA synthesis when tested in EUE cells (Benigni and Dogliotti, 1980a,b; Benigni et al., 1982). Rocchi et al. (1980) reported that triallate, at a concentration of 100 µg/mℓ, induced a 57% inhibition of DNA synthesis in rat thymocytes, a 52% inhibition of DNA synthesis in human lymphocytes and a 5% inhibition of unscheduled DNA synthesis in human lymphocytes.

In the dominant lethal assay, Johannsen et al. (1977) administered triallate to male mice at dose levels of 200 or 400 mg/kg via i.p. injection. Triallate gave a negative response under the conditions of this test system, as the occurrence of dominant lethal effects in the treated mice was similar to control values. The dominant lethal assay may not be sensitive to some chemical mutagens and consequently the negative response must not be construed as a reflection of nonmutagenicity.

Based on the weight of evidence approach, it may be concluded that triallate has the potential to be mutagenic. It induces reverse mutations in Salmonella typhimurium, forward mutations in Streptomyces coelicolor and Aspergillus nidulans, growth inhibition in Bacillus subtilis, mitotic recombination in Saccharomyces cerevisiae, gene mutations in mammalian cell cultures and unscheduled DNA synthesis in rat thymocytes and human lymphocytes. Triallate also induces chromosomal aberrations and SCEs in CHO cells.

5.3. TERATOGENICITY

In a teratogenicity study conducted at Monsanto Laboratories, Johannsen et al. (1977) administered triallate orally to pregnant rabbits at doses of 3 or 10 mg/kg on days 6-18 of gestation. Neither dose level induced a teratogenic response in the offspring of treated dams. Further details of this study, including the presence or absence of maternal toxicity and/or fetotoxicity, were not reported in the proceedings abstract, and no published paper on this study was found.

5.4. OTHER REPRODUCTIVE EFFECTS

In a multi-generation reproductive study that was in progress at Monsanto Laboratories, Johannsen et al. (1977) reported that no treatment-related effects were seen in rats fed triallate at dietary concentrations of 10, 30 or 100 mg/kg over two generations. No further details of this study were reported in the proceedings abstract, and no published paper regarding this study was found.

As reported in the abstract of a Russian study, Zhavoronkov et al. (1973a) administered triallate at 1/100 of the LD_{50} in the diet of hens for 10 days and observed resultant decreased fertility, impaired development of chicks and hemorrhaging and degenerative-necrotic changes in the liver, kidneys and reproductive organs. The abstract did not elaborate on the exact dose administered, the LD_{50} or the nature of the observed effects, severely limiting the usefulness of this study.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

In a 2-year chronic oral toxicity study conducted at Monsanto Laboratories, Johannsen et al. (1977) administered triallate to groups of 50 male and 50 female rats at dietary concentrations of 0, 50, 100 or 200 ppm.

Depressed weight gain was observed in females at the 200 ppm level; food consumption data were not reported. For all dose levels tested, there were no gross pathological changes and no effect on hematologic indices, clinical chemistry, urinalyses, absolute organ weights or organ weight ratios when compared with corresponding control values. Further details of this study were not reported in the proceedings abstract, and no published paper on this study was found.

The abstracts of two Russian subchronic toxicity studies provide scanty detail in regard to duration and number of exposures and observed effects, while information regarding controls and the number and sex of animals tested is not available. Rappoport and Pestova (1974) administered triallate repeatedly to rats at a dose level of 14.71 mg/kg (10% of the LD_{50}) for 1-4 months. The treated animals developed edema and plethora in the brain and other unspecified internal organs. Pestova (1968) exposed cats to atmospheric concentrations of triallate at a level of 5.3 mg/m^3 for 4 hours/day for $\geq$4 months. This level of exposure, reported to be the threshold for toxic action in cats, produced transient increased acetic acid concentrations and leukocyte counts during the first 2 months of exposure that returned to normal during the following 2 months of exposure. Effects of higher levels of exposure were not mentioned.

In the abstract of another Russian study, Voitenko et al. (1972) reported that continuous atmospheric concentrations of 2.7-4.6 mg/m^3 of triallate in the environment of farm workers resulted in decreased arterial pressure in exposed workers. The duration of exposure and other pertinent data were not reported in the abstract, and the paper was not available for review.

5.6. OTHER RELEVANT INFORMATION

Wiswesser (1976) lists oral LD_{50}s for triallate of 1675-2165 and >20,000 mg/kg for rats and dogs, respectively, and a dermal LD_{50} of 2225-4050 mg/kg for rabbits.

The acute toxicity of triallate to several species of laboratory and domestic animals has been documented in several Russian studies, of which only the abstracts were available. Pestova (1968) reported oral LD_{50} values for triallate of 930 and 1471 mg/kg for mice and rats, respectively; application of triallate to skin resulted in slight irritation (species and duration of exposure not reported). Rats administered triallate in a single oral dose of 1471 mg/kg (LD_{50}) were observed to have disrupted functioning of the cardiac, respiratory and nervous systems, whereas administration of 1000 mg/kg (MTD) resulted in decreased succinic dehydrogenase and lactic dehydrogenase activities, decreased hepatic thiol content and an increased hepatic pyruvic acid level. Voitenko et al. (1967) also administered a single oral MTD (assumed to be 1000 mg/kg) to rats and observed a 1.3- to 2.5-fold increase in RNAase activity of the liver and spleen 18 hours after administration and a disruption in normal thyroid gland function, indicated by the pattern of ^{131}I absorption. Following administration of triallate emulsions (dose levels not reported) to mice, rats and cats, Pestova (1966) observed tearing, salivating, panting and mobile excitement within 10-15 minutes, followed by depression and clonictonic convulsions. Single oral administration of triallate at LD_{50} levels of 930 mg/kg to mice and 1471 mg/kg to rats produced a decrease in cholinesterase activity in the liver, kidneys and lungs, and an accompanying increase in blood pyruvic acid content (Pestova, 1966).

Verkhovskii (1972) reported oral LD_{50} values for triallate of 500 and 945 mg/kg for rabbits and rats, respectively. A single oral administration of triallate to rabbits and rats at the respective LD_{50} levels resulted in decreased brain acetylcholinesterase activity and a decreased number of erythrocytes in peripheral blood. Verkhovskii (1972) also reported effects of parenchymatous cell hyperemia, brain hemorrhages, focal hepatic cell necrosis and spleen hyperplasia. Following oral administration (duration not reported) of triallate at a dose of 450-500 mg/kg (approximate LD_{50}) to rabbits, Zhavoronkov et al. (1973b) observed an inhibition of acetylcholinesterase activity in the peripheral blood and CNS, with the medulla oblongata reported to be the most sensitive part of the brain in regard to triallate exposure, and decreased osmotic resistance of erythrocytes in proportion to blood acetylcholinesterase inhibition.

Verkhovskii (1972) and Verkhovskii et al. (1973) administered triallate in a single oral dose of 300 mg/kg to sheep and pigs and observed resultant altered hematologic parameters, including decreased levels of RNA and DNA, an increased level of free nucleotides, transient changes in total plasma protein content, an increase in albumin and a decrease in the globulin fractions. Zhavoronkov and Verkhovskii (1975) observed an increase in total blood glutathione in sheep 8-10 days after administering triallate as a single oral dose of 300 mg/kg and a decrease in reduced glutathione and accompanying increase in oxidized glutathione in pigs 5 days after administering triallate as a single oral dose of 720 mg/kg. Both species were reported to have decreased blood peroxidase and increased blood sugar 8 days post-treatment.

Evodokimov et al. (1980) administered triallate to mice, chickens, rabbits, sheep and pigs (dose and route of administration not reported).

Following acute poisonings, postmortem examinations revealed congestive hyperemia, hemorrhages and serous edema in treated animals. Changes in parenchymatous organs were characterized by the development of dystrophy and inflammation of the gastrointestinal tract. Decreased egg production was observed in treated chickens within 3.5 days after treatment.

6. AQUATIC TOXICITY

6.1. ACUTE TOXICITY

Pertinent data regarding acute effects of triallate were not located in the available literature.

6.2. CHRONIC TOXICITY

Pertinent data regarding chronic effects of triallate were not located in the available literature.

6.3. PLANT EFFECTS

Kratky and Warren (1971) reported that 18-36 hours of incubation in 1 and 10 ppm triallate had no effect on growth of the alga, Chlorella pyrenoidosa.

6.4. RESIDUES

Pertinent data regarding triallate residues were not located in the available literature.

6.5. OTHER RELEVANT INFORMATION

Pertinent data regarding other relevant information on triallate were not located in the available literature.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

According to the Federal Register (FR), tolerances for triallate residues have been established under the Federal Food, Drug and Cosmetic Act at 0.05 ppm in or on canary grass seed and straw (47,196 FR 44563-44564) and in or on grain and straw of barley and wheat, lentils and lentil forage and hay, and peas and pea forage and hay (37,38 FR 3988-3989).

In the USSR, the recommended maximum permissible concentration for exposure to triallate in air is 1 mg/m^3 (Pestova, 1968).

7.2. AQUATIC

Guidelines or standards for the protection of aquatic organisms from the toxic effects of triallate have not been derived.

8. RISK ASSESSMENT

Triallate was tested for carcinogenicity by Johannsen et al. (1977). No carcinogenic effects were seen in rats administered triallate at dietary concentrations of 0, 50, 100 or 200 ppm for 2 years. The lack of sufficient detail reported in this proceedings abstract and the lack of a full paper on this study preclude the use of these data for quantitative risk assessment.

Positive results have been obtained for triallate in some *in vitro* mutagenicity assays with bacteria (Carere et al., 1978a,b; Sikka and Florcyzk, 1978; DeLorenzo et al., 1976, 1978; Douglas et al., 1981a,b; Sandhu et al., 1981; Kasica et al., 1981; Wildeman and Nazar, 1982; Shiau et al., 1981) and yeast (Morpurgo et al., 1977), but not *in vivo* tests with *Drosophila melanogaster* (Sandhu et al., 1981; Kasica et al., 1981). Other assays for genotoxicity in cultured rodent and human cells also have given positive results (Douglas et al., 1981a,b; Benigni and Dogliotti, 1980a,b; Benigni et al., 1982; Rocchi et al., 1980).

At dose levels of 3 or 10 mg/kg administered orally to pregnant rabbits on days 6-18 of gestation, triallate did not induce an increased incidence of terata (Johannsen et al., 1977). No treatment-related effects were seen in rats fed triallate at dietary concentrations of 10, 30 or 100 mg/kg over two generations (Johannsen et al., 1977).

Little information is available regarding the effects of chronic exposure to triallate. The chronic dietary study of Johannsen et al. (1977) appears to define a free-standing NOAEL of 200 ppm in rats; however, the available proceedings abstract did not provide adequate detail regarding the effects induced by triallate exposure, and no published paper on this study was found. The only other studies available that indicate a LOEL were

published in the Russian literature and were available only as abstracts. Pestova (1968) reported that exposure to 5.3 mg/m³ of triallate vapor for 4 hours/day was the threshold for toxic action in cats, producing transient increased acetic acid concentrations and leukocyte counts during the first 2 months of exposure, which returned to normal during the following 2 months of exposure. Voitenko et al. (1972) reported decreased arterial pressure in farm workers exposed to atmospheric concentrations of 2.7-4.6 mg/m³ of triallate. These data are not an adequate basis for the derivation of an ADI value for triallate.

Statements concerning available literature in this document refer to published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment can, however, be presented in this chapter.

The CBI Appendix to this document reviews other animal data from which an ADI of 0.015 mg/kg bw/day, or 1.05 mg/day for a 70 kg man, was derived. The ADI was based on a NOAEL of 15.0 mg/kg bw/day, which was near the threshold for clinical anemia in dogs in a 2-year subchronic dietary study and an uncertainty factor of 1000 (10 to account for interspecies extrapolation, 10 for protection of unusually sensitive individuals within the human population, and 10 for the use of a subchronic NOAEL for human ADI derivation). The CBI Appendix mentions a chronic oral study in rats, performed by Industrial Bio-Test for the Monsanto Company; this study was not reviewed in detail because it has been declared invalid (U.S. EPA, 1983).

9. REFERENCES

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APPENDIX

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
EPCASR
Chemical Industry Notes

Most of these searches were conducted in February, 1983; a few were conducted March-May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 7 and 8th), and the following secondary sources were reviewed:

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